

tality was 34 per cent. Three cases after recovery had marked mental disturbance and had to be treated in asylums. Four patients recovered from eclampsia without coming into labor, three of whom went to full term and one was delivered prematurely. In one case a full term, well-developed child developed eclampsia convulsions seventeen hours after birth, which proved fatal.

OTOLOGY

UNDER THE CHARGE OF

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The Pathology of Otosclerosis.—By J. S. FRASER and R. MUIR (*Jour. Laryngol., Rhinol., and Otol.*, vol. xxxi, No. 11).—This communication, a part of the report for the year 1915 from the ear and throat departments of the Royal Infirmary at Edinburgh, is a valuable contribution to the record of the pathological facts and an inferential study of the etiology of the several forms of tissue change in the middle ear or labyrinth, which have been grossly included under the title name of this communication, and includes various references to the literature of the subject and suggested explanations of the concomitant symptoms, the paper moreover being richly illustrated by plates of microscopic sections. The theories as to the pathology of otosclerosis fall under four headings: That it may be congenital; that it may follow inflammatory changes in the middle ear; that it may be due to infection through the blood; and that it may be caused by abnormal conditions in the nerve supply of the parts affected. (1) According to the first theory, otosclerosis is a congenital anomaly of the process of growth in the petrous portion of the temporal bone, due to the presence of certain determinants in the patient's blood. This anomaly only becomes manifest after puberty. (2) Otosclerosis is regarded by some as a chronic inflammatory process spreading from the mucoperiosteum of the middle ear and following attacks of catarrhal or suppurative otitis media. The inflammatory condition is supposed to linger about the niche of the oval window and to invade the bone from the deep layer of the mucosa. Such an invasion may be favored by the anastomosis which occurs at this spot between the tympanic vessels and those of the bony labyrinth capsule. Further, as has been pointed out, the joints in the middle ear are the only articulations in the body covered by mucous membrane, and therefore liable to infection from the surface. (3) A third group of otologists look on otosclerosis as a primary disease of the bone, the infection coming by the way of the blood-stream, as in osteomalacia, rheumatism, etc. Ferreri regards otosclerosis as closely allied to osteomalacia, and finds that almost all patients suffering from the latter disease are also the subjects of otosclerosis. A variant of this view is held by A. A. Gray, who has put forward the theory that otosclerosis is an asepti-

necrosis of certain areas in the labyrinth capsule—apparently as a result of aseptic infarction. Gray believes that similar areas occur in other bones of the skeleton. (4) Lastly otosclerosis is regarded by a small group of observers as due to a trophic disturbance—i. e., as a degenerative atrophic process. According to this theory the changes (sometimes) found in the ganglia and nerves are primary, while those in the labyrinth capsule are secondary. In this connection it may be noted that certain cases, which clinically appeared to be examples of *nerve deafness*, on microscopic examination showed spongification of the labyrinth capsule with (Siebenmann) or without (Bruehl) ankylosis of the stapes. This type of otosclerosis associated with nerve deafness—the so-called “atypical otosclerosis”—is markedly hereditary. The author of the second part of the paper gives a compact statement of the pathologic changes as determined under section and microscopic observation, and points to the fact that the study of sections from cases of otosclerosis strongly suggests the structural changes met with in other bony tissues in conditions of chronic toxemia or altered metabolism. Whatever be the exciting cause of this condition we have a series of progressive changes which appear to be *purely inflammatory* in character. The ultimate result is a typical and constant lesion, viz. marked sclerosis of the bony tissue of the labyrinth capsule. The alleged absence of plasma cells does not prove that otosclerosis is of non-inflammatory origin. These cells are not present in all forms of chronic inflammation. Even when they are present they do not occur in all stages of the inflammatory process. For their detection special staining methods are required—methods difficult to apply to celloidin sections of the labyrinth. The first change one observes in otosclerosis is the marked and general engorgement of the bloodvessels both in the fatty marrow of the bone and in the mucoperiosteum. The connective tissue of these areas is also seen to increase in amount, having a loose, open, and fibrillated structure. There are more cellular areas, mainly composed of mononuclear cells, which are closely related to the congested vessels. Some of the medullary spaces in the petrous bone show leukoblastic marrow reaction. Such appearances can be explained by a toxic body brought by the blood or stimulant, especially on the connective tissues of the marrow. The condition is essentially a granulating process, which slowly brings about absorption of the bone, allowing the opening up of the medullary spaces. The deeply staining bone seen around the enlarged medullary spaces appears to be old bone which is undergoing change, and not new-formed bone, as described by various writers. The granulation tissue tends to become more fully formed fibrous tissue, and these fibers range themselves in concentric layers around the central vessel. Ossification of this fibrous tissue now sets in, beginning with the fibers lying in close relation to the bone trabeculae, thus bringing about a marked thickening of the lamellae at the expense of the Haversian canals. The inner layer of the bone in the labyrinth capsule also appears to be involved in this process of sclerosis. There is an active proliferation of the cartilage cells and ossification into a bony matrix.

The above changes seem to be of a patchy character and appear to begin at the junction of the cartilaginous and lamellar portions of the labyrinth capsule, and to spread directly along that line for some distance, extending more or less into the cartilaginous and lamellar bone

on each side, and in places passing right through the cartilage bone to reach the endosteal tissue lining the cavities of the inner ear. This path of extension strongly suggests some direct channel, such as that shown to exist, and would explain the areas of change seen in the various parts of the labyrinth capsule. The whole process appears to be a slow form of inflammation, a definite series of reactions of a special tissue to repair damage caused by some unknown element, bacterial or toxic. As a result of their investigations the authors arrive at the following conclusions. (1) Some cases of otosclerosis (spongification of the labyrinth capsule) appear to be of the nature of a blood infection, and may be called "primary." We would expect that further research would show that in these cases the bony changes are widespread throughout the skeleton. (2) Other cases of otosclerosis undoubtedly follow attacks of catarrhal or purulent otitis media, the infective process invading the labyrinth capsule at the anterior margin of the oval window. Such cases may be called "secondary." (3) From the clinical stand-point, heredity plays an important part in otosclerosis, but, as the condition is a chronic inflammatory process, the infective agent must gain access to the labyrinth capsule either through the blood-stream or from the middle-ear cleft. (4) It has been proved by clinical and microscopic research that "atypical" cases of otosclerosis exist in which functional examination of the ear reveals the presence of nerve deafness. The relationship between the bony changes and the nervous affection is not yet clear.

HYGIENE AND PUBLIC HEALTH

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The Minimum Number of Bacilli Required to Transmit Tuberculosis to Guinea-pigs.—I. THÖNI and A. C. THAYSEN (*Zentralbl. f. Bakteriologie*, 1916, Part 1, p. 308) report the following: The dose of injection was standardized by Burri's method, which permits an accurate count of the bacteria. In the first series of experiments 19 guinea-pigs were injected with a highly pathogenic culture, the individual doses of which contained between 10 to 76 tubercle bacilli. Only one pig, the one which had been injected with 71 bacilli and which died after forty-one days with symptoms of dysentery, showed signs of tuberculosis. In a second series of experiments 3 strains of the bacillus, two of which were highly virulent, were employed. Twenty-two guinea-pigs received doses containing, between 99 and 343 bacilli. No tuberculosis developed.